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Modification of Physiological and Subjective Responses to Stress through Heart Rate Biofeedback

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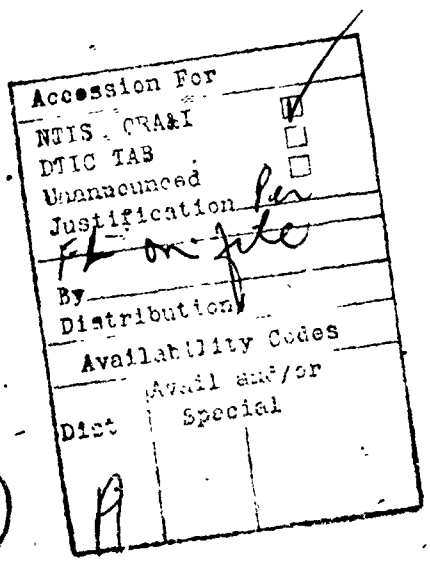

Biofeedback research is now more than 15 years old, and considerable progress has been made in dealing with theoretical and empirical questions about the processes involved. The widespread emphasis on clinical applications, however, has tended to divert attention away from more systematic basic research on biofeedback (see Black, Cott, & Pavloski, 1977; Gatchel & Price, 1979; Shapiro, 1980). As a means of examining some of the mechanisms of biofeedback learning and of developing new ways of applying biofeedback clinically, the research to be discussed in this chapter poses different questions than have been asked in the past and utilizes a different experimental paradigm.

Typically in biofeedback, the individual's ability to modify physiological responses is evaluated under resting conditions. Feedback and reinforcement are given for spontaneously occurring responses, and no external stimuli are presented other than the tones, lights, or meters that make up the biofeedback displays. No demands are placed on the individual other than those of the biofeedback task itself. The strategy employed in the present research is to present stimuli to the individual which elicit specific physiological responses, or changes in physiological arousal, and to determine whether such elicited physiological responses, or related anticipatory physiological responses, can be modified by means of biofeedback training procedures. Electric shock to the forearm and immersion of the hand in ice water were used as a means of arousing physiological and emotional responses.

A second aim of the research was to determine whether perceptions of the intensity of such stressful stimuli or other subjective reactions would also be modified as a consequence of the biofeedback training.

This paradigm enabled us to investigate the adaptive or psychological significance of specific physiological component responses associated with emotional arousal and reactions to stress.

The use of biofeedback to alter a specific physiological response and thereby affect emotional arousal can be compared with the use of autonomic nervous system drugs for similar purposes. For example, a beta-adrenergic blocking agent, propranolol, has been used in research on the modification of anxiety in chronically anxious patients (Tyrer, 1976). The aim was to determine if reductions in beta-adrenergic functions, such as a reduction in heart rate, produced by the drug would be associated with reductions in anxiety. Tyrer found that propranolol led to reductions of anxiety, particularly in patients who report normally experiencing their anxiety in somatic or bodily terms. Biofeedback provides a behavioral means of studying similar processes. In the present research, the biofeedback procedure was oriented directly to the control of physiological responses which are part of the individual's total reaction to specific stressful stimuli with the idea that such techniques may be useful to therapists in the management of anxieties, fears, and phobic reactions to such stimuli. Voluntary control of autonomic functions facilitated by biofeedback methods may be a useful strategy in the treatment of stress-related disorders. To the extent that physical symptoms are under the control of specific stress-related stimuli, then it would be useful to adapt biofeedback training methods which involve the appropriate stimuli. For example, biofeedback training procedures could be adapted to help patients with Raynaud's disease reduce their abnormal vascular response to cold temperatures or to specific emotional or other triggering stimuli. Rather than having the biofeedback training occur in resting conditions, the task for the patient would be to

increase skin temperature or blood flow while the stress-related stimuli are presented.

Two additional questions provided further impetus for the research to be described in this chapter. (1) We wanted to make a direct comparison of biofeedback effects under resting and stress conditions. Would it be more or less difficult for subjects to modify specific physiological responses associated with aversive stimuli or stress as compared with non-stress conditions? (2) Reductions in arousal-like activity (decrease in heart rate and blood pressure, increases in skin temperature) have been difficult to demonstrate in biofeedback studies. Would it be easier to obtain decreases in arousal when tonic levels are heightened under stressful conditions?

Control of Elicited Electrodermal Responses

An empirical justification for the use of biofeedback as a means of altering elicited physiological responses to stressful stimuli derives in part from earlier studies in our laboratory on the control of electrodermal responses elicited by simple non-aversive stimuli. Shnidman (1970) examined the extent to which the skin potential response elicited by presentation of a small red triangle for 5-sec periods could be instrumentally conditioned. One group of subjects was reinforced each time they showed a criterion skin potential response to the stimulus. A second group was reinforced on the same trials as experimental subjects with whom they were matched for electrodermal responsivity, whether they responded or not. The reinforcers were slides of interesting landscapes and animals, equated with monetary bonuses. Significantly more skin potential responses were shown in the experimental than in the control group. In related experiments, avoidance, punishment, and other conditioning procedures yielded further supporting evidence that

electrodermal responses elicited by simple stimuli can be shaped instrumentally (Grings & Carlin, 1966; Kimmel & Baxter, 1964; Shnidman, 1969; Shnidman & Shapiro, 1971). Shnidman (1970) concluded, "The goal of desensitization is the modification of autonomic responses to specific stimuli and situations. Monitoring and conditioning autonomic responses to critical stimuli may effect desired changes in the autonomic activity and behavior" (p. 494). This anticipated the work to follow.

An unpublished pilot study from our laboratory attempted to follow this lead (Shapiro, Schwartz, Nelson, Shnidman, & Silverman, 1972). Volunteer subjects reporting moderate to intense fear of snakes were shown slides of snakes in order of increasing "fear" quality. Each slide was presented for 5 sec. Half the subjects were reinforced whenever the skin resistance response elicited by a slide was larger than for the previous slide, and half the subjects were reinforced for smaller electrodermal responses. Both groups tended to show habituation in their elicited electrodermal responses, but the rate of habituation was greater in the decrease group. Before and after the conditioning trials, a snake and spider fear questionnaire was administered to subjects. Both groups showed a reduction in expressed fear, but the reduction was greater in the decrease group.

Mention should be made here of one of the first studies attempting to modify a physiological response as a means of modifying associated performance or behavior--in this case problem solving and cognitive functioning (Kimmel, Pendergrass, & Kimmel, 1967). Children were reinforced with candy and approval when they showed decreases in skin resistance on the presentation of geometric stimuli used in the Seguin form board task. It was found that the elicited electrodermal responses could be modified in this fashion.

Moreover, although the results were quite complex, the conditioning appeared to transfer to the children's form board performance in a subsequent task (placing the forms in their proper places). Children reinforced for "orienting" did better on this "intelligence" test than those reinforced for not so responding.

For a further discussion of related biofeedback research on electrodermal, electromyographic, and electroencephalographic responses associated with emotional behavior, see McCroskery and Engel (1979). This paper will focus on the control of heart rate responses and its consequences for behavior.

Control of Anticipatory Heart Rate Responses

Heart rate was chosen for feedback training in the stress paradigm because of the ease with which subjects can learn voluntary control of this function (Brener & Hothersall, 1966; Engel & Hansen, 1966; Lang, 1974; Shapiro, Tursky, & Schwartz, 1970) and the oft-demonstrated empirical association between heart rate and fear or anxiety (Lang, Melamed, & Hart, 1970; Lang, Rice, & Sternbach, 1972).

Although psychophysiological research has closely linked together heart rate and emotional stimulation, it is not known whether heart rate merely reflects emotional responding or actually plays a more direct role in modulating the relative degree of such responding. DiCara and Weiss (1969) reported that curarized rats receiving operant training for heart rate increases demonstrated a deficit in subsequent skeletal shock avoidance and escape learning in the noncurarized state, when compared to rats given prior operant heart rate slowing training. Avoidance and escape performance was directly related to emotionality. Rats exhibited much more emotionality, as indexed by jumping, turning, and freezing behaviors after operant training in heart rate speeding and less emotionality after operant heart rate slowing.

A second experiment was reported in the DiCara and Weiss study on the degree to which the skeletal-avoidance learning was affected by shock intensity. It was hypothesized that the avoidance learning would become progressively disrupted at higher shock intensity levels. The results supported this hypothesis and suggested that "learning to speed up heart rate in a shock-avoidance situation had the effect of increasing fear or excitability" (p. 372). The mechanism by which heart rate increase mediated fear (and poor avoidance learning) is uncertain. The authors proposed that fast heart rate learning produced strong fear which led to unconditioned competing responses that interfered with avoidance learning. No data were presented concerning concurrent changes in other physiological responses and the degree to which the heart rate effects were specific or were associated with an overall physiological arousal pattern. In a related study in monkeys, classically conditioned changes in heart rate occurring in anticipation of electric shock could be significantly altered by operant reinforcement (Ainslie & Engel, 1974).

We examined the ability of human subjects to alter their heart rate in anticipation of receiving aversive stimuli. The first experiment (Sirota, Schwartz, & Shapiro, 1974) consisted of 72 15-sec trial periods, half followed by 2 sec of aversive electric shock stimulation to the forearm. Different colored lights remaining on for each 15-sec period signaled whether shock would follow or not. There were two groups of subjects (n = 10 per group). The first was instructed to increase heart rate and was given cardiotachometer feedback plus monetary bonuses for criterion heart rate increases. The second group was a heart rate decrease condition. Following each shock trial, subjects were asked to rate the intensity or painfulness of the stimulus on a 100-point scale. Significant differences both in tonic

heart rate (heart rate per trial) and in phasic heart rate (change in heart rate during the 15-sec periods) were obtained between the two groups. The magnitude of the heart rate effects achieved was as large or larger than reported in typical biofeedback studies not involving specific eliciting stimuli. By and large, heart rate control was not interfered with when subjects were expecting to be shocked, compared with safe periods. In fact, rather than being disruptive of heart rate control, anticipation of shock seemed to result in larger bidirectional differences in phasic heart rate changes during the periods of anticipation.

Subjects in the increase group rated the shocks as more intense than subjects in the decrease group. The difference was present in the early trials and did not change appreciably over the course of training. A separate analysis of "cardiac aware" versus "cardiac unaware" was undertaken, this variable defined by subjects' responses on an autonomic perception questionnaire adapted from Mandler, Mandler, and Uviller (1958) in which subjects were asked to indicate their awareness of physiological changes during fear situations in daily life. Two items dealing specifically with cardiac functioning--loud pounding heart, increase in heart rate--defined the cardiac awareness dimension. Cardiac aware subjects rated these items as highly relevant to their fear. Reanalyzing the results in terms of this dimension, we found that cardiac aware subjects in the increase group rated shocks as more and more intense over the course of training; cardiac aware subjects in the decrease group tended to rate the shocks as less intense over trials. No differences between increase and decrease feedback conditions were obtained for the unaware subjects. These cardiac aware-unaware findings parallel Tyrer's (1976) results using pharmacological control of heart rate in somatically-oriented anxious patients. Inasmuch as a

no-feedback control group was not run in this study, there is no way to determine whether the biofeedback procedure served to facilitate a decrease in heart rate under the heightened conditions of arousal in this experimental situation. Nor were data available on associated physiological changes or respiration.

To replicate and extend these findings, Sirota, Schwartz, and Shapiro (1976) did an experiment which was divided into two parts each consisting of a baseline period and 25 15-sec heart rate biofeedback trials. Four out of five trials ended with the presentation of an aversive electric shock. One group of subjects was instructed to increase heart rate during the first part of the experiment and decrease it during the second part. A second group of subjects was studied in the reverse order (n = 10 per group). Subjects were successful in increasing and decreasing their heart rate in anticipation of the aversive electric shock stimulation. In addition, the increases and decreases in heart rate were generally associated with parallel changes in subjective ratings of painfulness of the stimuli, expecially in cardiac aware subjects. In this experiment, heart rate was also measured during "blank trial" periods occurring between trials to assess whether bidirectional control was being achieved relative to non-specific changes in heart rate over time. Generally, heart rate during increase trials was higher, and heart rate during decrease trials was lower, as compared with these control periods. The heightened arousal of this experimental situation may have helped demonstrate this bidirectional effect. No data were available on other physiological measures or respiration.

In this connection, DeGood and Adams (1976) compared the relative effectiveness of biofeedback for heart rate decreases, progressive relaxation, and a non-contingent music control group. Following an initial

series of shock trials to determine pain levels, subjects received 25 min of training, and then were instructed to lower their heart rate during 10 tone-shock pairings. Reliable heart rate reductions were found during the tone-shock pairings for the biofeedback and progressive relaxation groups. Reliable reductions were also reported in pre-to-post state anxiety and shock ratings, but no group differences emerged. Other techniques such as relaxation may be as effective as biofeedback in decreasing emotional arousal in this situation.

Taken together, the results support the notion that learned control of heart reactions to aversive stimulation may result in associated changes in subjective pain ratings. However, the actual role heart rate plays in affecting such perceptual changes remained unclear in light of evidence indicating that merely the belief that cardiovascular changes are occurring can result in changes in avoidance behavior and reports of pain (Borkovec, 1976; Holmes & Frost, 1976; Valins & Ray, 1967). It is conceivable that the belief that heart rate is increasing or decreasing may be sufficient in itself to alter subjective ratings of pain, particularly in cardiac aware subjects. Moreover, the advantages of biofeedback over other methods of self-regulation remains an open question.

In an attempt to disentangle the relative effects of cognitive factors and heart rate on changes observed in subjective pain ratings, Sirota (1976) examined several unique combinations of hea[illegible].ate feedback and instructions using a similar anticipatory shock paradigm. Four groups of cardiac aware subjects were studied (n = 10 per group). Group 1 was given instructions to increase heart rate during Part 1 of the experiment and to decrease heart rate during Part 2 and provided with veridical heart rate feedback during both parts. This group was intended to replicate their previous

findings. Group 2 was given instructions to increase heart rate during Part 1 and then to decrease heart rate during Part 2, but given feedback for heart rate stabilization in both parts. The purpose of this group was to make subjects believe that heart rate was changing while in fact it remained constant. Group 3 was given instructions to stabilize heart rate during Parts 1 and 2, but given feedback for increasing heart rate during Part 1 and decreasing it during Part 2. This attempted to instill the belief that heart rate was not changing while in fact it was. Group 4 was given instructions to stabilize heart rate and provided with veridical feedback during both parts of the experiment. The results of this experiment were complicated since expected heart rate changes were not clearly obtained. Generally, subjects found it difficult to do one thing with their heart rate after being instructed to do another. A comparison of group heart rate changes suggested the prepotence of instructions over feedback in the control of heart rate with group shock ratings tending to parallel heart rate, particularly in Part 1. These results lent further support to the conclusion that a combination of physiological and cognitive factors is required for a learned heart rate response to transfer to a related perceptual change. The question of cognitive-physiological interaction will be taken up again below.

Control of Heart Rate Response to Cold Pressor Stress

The previous studies required subjects to control their heart rate in anticipation of an aversive stimulus. More recently, our research has focused on the ability of subjects to control their heart rate while actually experiencing aversive stimulation. Victor, Mainardi, and Shapiro (1978) investigated the effects of biofeedback training on heart rate and subjective reactions to the cold pressor test--immersion of the hand in ice water for

30 sec. The cold pressor test was chosen because it elicits reactions that are predictable, including tachycardia and pain in most human subjects. In addition, the test-retest reliability for heart rate changes and pain reports is high (Hilgard, 1975; Hilgard, Morgan, Lange, Lenox, Macdonald, Marshall, & Sachs, 1974; Lovallo, 1975). Following an initial 30-sec cold pressor test, subjects were assigned to one of five experimental conditions (n = 9 per group): 1) meter biofeedback for heart rate increase; 2) meter biofeedback for heart rate decrease; 3) instructions to increase heart rate with no feedback; 4) instructions to decrease heart rate with no feedback; and 5) a habituation control group (no heart rate instructions or feedback). A second 30-sec cold pressor test was given after 25 30-sec trials of training. Except for the habituation control condition, all groups were instructed to continue controlling their heart rate in the instructed direction during the second cold pressor test. No feedback was given. A summary of the pain rating and heart rate effects is given in Table 1. Subjects in the feedback groups exhibited reliable heart rate increases and decreases as well as reporting parallel changes in subjective pain ratings. The no-feedback groups showed similar heart rate and pain rating trends, but the differences failed to reach statistical significance. In this study, cardiac awareness was not significantly correlated with cold pressor pain ratings (Shapiro, 1977).

Given a single session of biofeedback training, subjects were able to gain voluntary bidirectional control of heart rate while being subjected to the noxious stimulation of ice water. Inasmuch as the biofeedback training itself was carried out in ordinary resting-condition trials, the results indicate that the effects of such training can carry over to a stress situation. Moreover, it may be easier to demonstrate a decrease in arousal-like

physiological activity with biofeedback training, in this case for heart rate, when it is assessed under stress conditions. In any case, the learned control was not interfered with.

Table 1 shows that the differences in pain rating generally paralleled the differences in heart rate observed during the second cold pressor test; the higher the heart rate, the higher the report of painfulness. Of the five groups, only the decrease-feedback group showed a significant correlation between pain rating and heart rate and change in heart rate during cold pressor test 2; the larger the reduction in heart rate, the lower the pain ratings in these subjects. Again, changes in other physiological measures or respiration were not available in this study.

In the next study, Reeves, Shapiro, and Cobb (1980) attempted to replicate the basic findings in the Victor et al. (1978) cold pressor experiment while at the same time further exploring the interaction of instructional and physiological variables in affecting subjective ratings of pain. The study undertook to clarify further the relative contribution of changes in heart rate (by means of biofeedback training) and cognitive factors (instructionally-induced belief that heart rate is changing in a specified direction) in affecting subjective reports of pain during aversive ice water stimulation. Four experimental conditions were studied (n = 10 per group). Two conditions were essentially the same as the feedback conditions used in Victor et al. (1978) and were an attempt to replicate their findings of parallel changes in heart rate and pain during the cold pressor test. Group I-I was instructed to increase their heart rate and given veridical feedback; Group D-D was instructed to decrease their heart rate and given veridical feedback. In the other two conditions, subjects were also instructed either to increase or decrease their heart rate but the feedback display was

reversed: Group I-D was instructed to increase their heart rate but actually given feedback for heart rate decrease; Group D-I was instructed to decrease heart rate but given feedback for heart rate increase. Thus Groups I-D and D-I were led to believe that their heart rate was changing in the instructed direction, but an attempt was made to change it in the opposite direction through reverse biofeedback.

Following a 10-min resting baseline, a 45-sec anticipation period immediately preceded a 45-sec cold pressor test. Subjects verbally reported numerical pain values (0-10, open at the top) three times (each 15 sec) during both anticipation and cold pressor periods. Visual Analog Scales and psychophysically scaled descriptors were used to assess maximum pain, pain intensity, and reactivity. A second 3-min baseline was taken, and then 25 subject-initiated biofeedback training trials were given. Each trial consisted of a 15-sec no-feedback no-control pretrial followed by 45 sec of visual feedback. Following feedback training, another 3-min baseline was recorded and the final anticipation and cold pressor test was administered. Subjects were instructed to control their heart rate in the same direction as instructed during training, while immersing their hand in the circulating ice water, but without the aid of feedback. Pain ratings were again taken.

Several methodological differences between this study and the Victor et al. study were introduced. 1) The cold pressor stimulation was made more consistent and aversive by installing a circulating pump in the water in order to maintain a constant 0.5° C water temperature. The length of the cold pressor test was also extended from 30 sec to 45 sec and was preceded by a 45-sec signaled anticipation period. 2) A computer graphics display was used to present heart period feedback. The visual feedback was presented

In the form of a vertical feedback line the height of which was linearly related to the time in milliseconds between successive R-waves in the electrocardiogram. Each succeeding R-R interval generated a feedback line which appeared at equal intervals to the right of the preceding line on the display. The feedback lines remained on the GT display throughout each 45-sec training trial. The subject was therefore able to observe a history of his heart beat performance during each trial. Subjects were asked to try to make the vertical feedback lines as long or as short as possible, depending upon the condition. In order to control for individual heart rate variability, the display parameters and reward criteria were individualized for each subject. To equate for task difficulty, the display parameters for the increase and decrease feedback conditions were made equivalent in terms of the expected magnitude of heart rate effect. 3) A more precise attempt was made to accurately scale the subjects pain ratings. In the previous studies, pain was verbally reported using a 0-10 numerical scale. In this study, subjects were required to report their pain using several different scales. Subjects verbally reported their pain levels when signaled by the computer three times (every 15 sec) during the cold pressor. The pain scale ranged from 0 = no pain to 10 = intense pain. Numbers larger than 10 were permitted to be used for pain which increased beyond "intense." Thus, changes in pain during the cold pressor could be observed. This "open-ended" scale is similar to that previously used by Hilgard et al. (1974). In addition, an attempt was made to distinguish between the intensity (sensory) and affective components of the pain experience. Immediately following each cold pressor test, two lists of 15 psychophysically scaled pain descriptors, intended to assess (a) intensity component--how much the pain hurts, and (b) affective component--how the pain

feels, were presented. The subject chose the one word from each list of randomly orde ! descriptors that best described his maximum experience during the cold pressor tests (Gracely, McGrath, & Dubner, 1976). Since Gracely et al. calculated bias-free scale values for each descriptor using cross modality scaling, numerical values could be recorded for the purposes of analysis. Table 2 shows the descriptors and corresponding psychophysically scaled values. Visual Analog Scale (VAS) was also given after each cold pressor. A VAS is a 10 cm, horizontal, straight line, the ends of which are anchored by the extreme limits of the sensation or response to be measured (Scott & Huskisson, 1976). The VAS was anchored by "no pain" on the left end and "pain as bad as it could be" on the right end of the line. Subjects were instructed to place a vertical "hash mark" somewhere on the line indicating their maximum experience during the cold pressor test. The position of the hash mark was measured in centimeters to yield a pain score.

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The main results of the study are summarized in Table 3. For ease of presentation, the data will be discussed in terms of heart rate rather than heart period. Biofeedback training resulted in heart rate increases for Group I-I and small heart rate decreases for Groups D-D, I-D, and D-I. These heart rate changes seemed to be paralleled by concomitant changes in frontal EMG, respiration period, and inspiration time, implicating possible somatic influences on heart rate changes during biofeedback training. The biofeedback data do not support the notion that instructions, and not biofeedback, are solely responsible for heart rate changes since Groups I-D and D-I failed to produce substantial heart rate changes in the instructed direction. The cold pressor results showed that subjects can increase and decrease their heart rate during painful stimulation following heart rate biofeedback training. Group I-I for whom instructions and feedback were veridical showed

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substantial increases in heart rate during the second cold pressor, as compared with Group I-D who was also instructed to increase heart rate but was given decrease biofeedback training. However, both groups instructed to decrease their heart rate showed comparable heart rate reductions, regardless of the direction of their prior biofeedback training. With the exception of Group D-I who showed marked increases in respiration period and inspiration time, no other concomitant physiological changes were observed during the final cold pressor.

When feedback and instructions were veridical, reliable changes in verbal pain ratings and the Visual Analog Scales were found during the cold pressors. Group I-I increased and Group D-D decreased their pain ratings from the first to the second cold pressor. Groups I-D and D-I did not show changes in pain reports. No differences were found for the intensity and affective scales although trends similar to VAS and verbal scales were found. Perhaps more sensitive measures of the intensity and affective components of pain will help determine whether self-regulated heart rate changes during the cold pressor alter pain through arousal (affective) mechanisms or through actual sensory (intensity) threshold changes.

Correlational analysis indicated that changes in pain perception are associated with heart rate changes during the cold pressor in the veridical conditions, especially in Group I-I ($r = 0.81$) and to a lesser extent Group D-D ($r = 0.68$). Correlations also showed pain perception to relate to the magnitude of heart rate change during biofeedback training for Group I-I ($r = 0.82$). Data from the groups given feedback opposite to instructions suggest that the pain perception effects are not a function of instructions per se (belief that heart rate is changing in the instructed direction). When instructions and feedback are opposite, pain ratings do not appear to depend on instructions or

on heart rate changes observed during the cold pressor test. Group D-I effectively reduced their heart rate response from the first to the second cold pressor by significantly slowing their respiration rate. However, Group D-I did not exhibit parallel changes in pain perception. A recent dissertation by J. D. Lane (1979) may provide an answer. Lane's dissertation showed that deliberately increasing or decreasing respiration rate during the cold pressor results in parallel increases and decreases in heart rate but does not affect pain perception. Thus, a subject may be able to voluntarily alter elicited heart rate responses in a variety of ways, but not all of these will necessarily result in associated behavioral or subjective changes.

The previous studies by Sirota et al. us. a shock stimulus showed the importance of cardiac awareness in predicting changes in pain reports. This study along with the Victor et al. study used the cold pressor test and failed to find a reliable relationship between cardiac awareness and pain. These results are perplexing but consistent, and possibly point to fundamental differences in the mechanisms underlying or mediating behavioral and subjective reactions to different laboratory stressors.

These data coupled with a previous pilot study reporting similar results (Reeves, Shapiro, & Cobb, 1979) suggest that a combination of veridical instructions and feedback are necessary for heart rate control during biofeedback and pain perception changes during cold pressor stimulation. At least for the veridical conditions, heart rate changes during biofeedback may be related to changes in pain perception. Our previous research has not determined whether these heart rate and pain perception changes are a function of the subject's ability to actually control phasic heart rate during cold pressor stress or whether the changes reflect a more tonic

shift in heart rate reactivity following biofeedback training. The final (Reeves & Shapiro, in press) study proposed to clarify this issue by giving veridical heart rate biofeedback for increasing (Group I) and decreasing (Group D) heart rate, and then testing heart rate reactivity and pain perception during cold pressor stress. All procedures in this study were identical to the previous study (Reeves et al., 1980) except subjects were specifically instructed not to alter their physiological reactions during the second cold pressor stress, rather to focus on accurately and honestly reporting their pain experiences. The results indicated that Group I increased their heart rate and Group D decreased their heart rate during biofeedback. Frontal EMG but not skin conductance showed a similar and reliable pattern. Both groups showed reliable reductions in heart rate, frontal EMG and skin conductance from the first to the second anticipation and cold pressor stress, with no reliable effects involving groups found. No group differences in pain ratings were found. Both groups showed a reliable increase in pain ratings on the second cold pressor stress. These data suggest that single session heart rate biofeedback training under resting conditions does not by itself alter subjects' heart rate reactivity to cold pressor stress. The previously reported differential control of heart rate during cold pressor stress probably reflects an acquired ability to alter phasic heart rate during cold pressor stress and not simply an alteration in tonic reactivity related to an overall change in physiological arousal.

Finally, a recently completed study conducted by Walter Greenberg provides further evidence regarding the functional significance of heart rate for perception of cold pressor pain. This study employed the same experimental paradigm as Reeves and Shapiro (in press) except that feedback was

given for systolic blood pressure rather than heart rate. A beat-to-beat tracking-cuff method of measuring blood pressure was used to provide feedback (Shapiro, Greenstadt, Lane, & Rubinstein, in press). Two groups were run, an increase and a decrease blood pressure condition (n = 10 per group). The hypothesis that learned changes in blood pressure would be facilitated under stress conditions received partial support. Of interest to this discussion was the finding that heart rate varied along with blood pressure during the feedback trials, relatively increasing for the increase group and decreasing for the decrease group. In the final cold pressor test, however, both groups showed a comparable decrease in heart rate as compared to their initial cold pressor response. Thus, the specificity of blood pressure training effects, at least with respect to heart rate, did not become apparent until the subject was placed under the cold pressor stress. Similar results were obtained in the Reeves et al. (1980) study. That is, EMG and respiration tended to follow heart rate during biofeedback trials, but during the final cold pressor only heart rate showed reliable changes in Groups I-I and D-D. As to pain perception, only one of six measures (reactivity) showed a significant effect (higher for the increase group). Assuming that the increase and decrease blood pressure instructions are comparable in their "emotional" implications to those used in the earlier heart rate research, these results lend support to the hypothesis that heart biofeedback rate/(with appropriate instructions) may be critical to the repeatedly observed pain perception effects occurring after heart rate biofeedback training.

Discussion

This chapter has described a program of research on the use of biofeedback techniques to augment or reduce heart rate changes occurring in antici-

pation of aversive electrical stimulation or in response to the painful stimulation of the cold pressor test. These laboratory stressors were chosen because they elicit relatively consistent increases in physiological and emotional arousal. The experiments were intended to provide a laboratory analogue of the behavioral control of pain, fear, and acute anxiety. Pain and fear are seen as complex patterns of physiological responses, overt actions, and various cognitive processes as indexed by verbal reports. The experiments attempted to demonstrate the utility of biofeedback methods as a means of selectively modifying physiological components of response to the two laboratory stressors and the effects of such modification on the individual's appraisal of the intensity or painfulness of the stimuli. The research was seen as a means of elucidating interactions between cognitive and physiological processes under conditions of stress and emotional arousal, and experiments were conducted in which the relative contributions of these two classes of events were evaluated.

The two sets of experiments described in this chapter involved control of heart rate in anticipation of electric shock and control of heart rate in response to the cold pressor test. By and large, similar trends, and hypotheses for further study, emerged from the two kinds of experiments. The cold pressor design appears to have certain advantages over the one used in the electric shock studies and will be emphasized in this discussion. The design involved an initial assessment of the individual's physiological and subjective responses to the stressor prior to feedback training as well as a reassessment of the same responses after the intervening period of feedback training. During the training, instructions and feedback were manipulated independently. Moreover, with this design, feedback can be given for variables other than heart rate to examine the adaptive significance of one function over another in the control of perception of pain or reports of fear or

anxiety. This last strategy has not as yet been explored extensively in our research. The biofeedback training was carried out under non-stress conditions, and the aim was to determine whether the training could transfer effectively to the condition in which the stress was presented.

Now to summarize the major findings. There is no question that the heart rate acceleration normally associated with response to immersion of the hand in ice water can be potentiated by means of heart rate biofeedback training combined with appropriate instructions. For example, in Victor et al. (1978), the increase in heart rate after feedback training was almost three times greater than prior to training. Subjects not given such training and simply instructed to increase their heart rate during the ice water immersion also potentiated their heart rate response but to a much lesser degree. The same is true for similarly instructed subjects who were given prior heart rate decrease training (even though instructed to increase their heart rate)(Reeves et al., 1980). Moreover, subjects given appropriate feedback training but instructed not to change their heart rate did not show an augmentation of their heart rate response (Reeves & Shapiro, in press).

Evidence has also been presented that biofeedback training methods can be used to attenuate the heart rate response to ice water stress. It is not clear, however, that biofeedback offered any special advantage over simple instructions to reduce heart rate (Reeves et al., 1980; Victor et al., 1978; see also Rupert & Holmes, 1978). Biofeedback is basically an active problem-solving procedure, involving information processing and presentations of stimuli and reinforcers which are arousing in and of themselves. In non-demanding conditions, heart rate may readily decelerate. Not trying actively to do anything or to achieve goals or rewards may be a good way to reduce physiological arousal. Subjects instructed to lower their heart rate during

the cold pressor test, even though they were given prior increase feedback training (without their explicit knowledge), seemed to be able to decelerate their heart rate (Reeves et al., 1980). Subjects given either increase or decrease feedback training but instructed not to change their heart rate actually reduced their tonic heart rate and to an equivalent degree when given the cold water stimulation. Their phasic response to the stimulus was apparently not affected. Subjects given no special instructions (Victor et al., 1978), a no-treatment control, showed little or no change in their heart rate.

Thus, training with appropriate instructions and feedback can effectively transfer to a stress condition, providing the individual a means of augmenting or reducing his normal response to the stress. It requires an active attempt on the part of the individual to apply the skill learned from the prior training. It is clear that prior appropriate biofeedback training plus the use of the acquired skill can facilitate a potentiation of heart rate when the individual is put under stress. In the case of response attenuation, however, an active attempt to reduce heart rate may lead to the desired result, regardless of the direction of prior feedback training. Such a reduction may be accomplished primarily through respiratory control, rather than being associated with a particular learned skill. Therefore, the experimental paradigm seems to offer an additional means of differentiating mediational processes involved in the voluntary control of physiological functions. This is supported by further evidence on the differential patterning of physiological changes that occurs during biofeedback training and during the stress transfer trials.

Although biofeedback training appears to offer advantages over simply instructing subjects to alter their heart rate, various forms of relaxation,

hypnosis, suggestion, mediation, and coping self-statements may also be effective in modifying physiological and emotional arousal (Benson, 1975; Chaves & Barber, 1974; Davidson & Schwartz, 1976; Goleman & Schwartz, 1976; Grimm & Kanfer, 1976; Hilgard, 1977; Meichenbaum, 1977; Reeves, 1976). Biofeedback techniques are primarily oriented to selective control of specific individual responses, and it seems more likely that such specificity will evolve as a result of biofeedback training than from such other procedures. If we can determine which physiological systems in an individual are particularly relevant to physiological and emotional arousal under conditions of stress, then it may be possible to tailor the feedback procedure accordingly.

When we turn to the pain perception data, complex interrelationships of cognitions and physiological changes become evident. The strategy of altering a physiological component of the individual's reaction to a stressful stimulus appears effective in altering reports of its painfulness. The perceptual effects depend on appropriate instructions as well as on the individual making a deliberate attempt to control his reactions on the basis of the instructions and prior feedback training. A reduction in tonic heart rate has no necessary consequence in and of itself for the individual's subjective response to stress, as is indicated by the results of Reeves and Shapiro (in press). The combination of appropriate instructions and veridical feedback is necessary for heart rate biofeedback training to exert a significant influence on pain perception.

One implication is that biofeedback or relaxation training for the purpose of reducing physiological arousal has no necessary effects on emotional reactions to stress or on anxiety (see Rupert & Holmes, 1978). Such decreased arousal has to be utilized by the individual as a deliberate and active coping skill, and the training has to be made directly relevant to the con-

ditions eliciting the anxiety or emotionality.

Finally, comments are in order on the adaptive significance of heart rate for pain and stress reactivity. In general, we found some association between heart rate and subjective response to stress--increase associated with increased emotionality, decrease with decreased emotionality, especially when appropriate instructions and feedback were coupled. Additional research will have to be carried out comparing the effects of feedback training for other physiological variables. McCroskery and Engel (in press) reviewed related research concerning the effects of electromyographic and electroencephalographic biofeedback training as a means of coping with stress. The research to date has been inconclusive. The difficulties and complications that beset research in EMG and EEG biofeedback in relation to emotional behavior also concern the research we have described in this chapter. The significance of a biofeedback strategy for elucidating cognitive and emotional processes depends on knowledge of what physiological responses or patterns of responses are related to the particular psychological state and the reliability of such a relationship. Moreover, the target state or behavior has to be reliably assessed. The choice of heart rate in our work does not eliminate some of the complex issues of interpretation. Our research focused on heart rate increases associated with presentation of an aversive stimulus. But it is well known that heart rate also increases during mental effort, positive emotional behaviors, feeding, and exercise. The advantage of the strategy described in this chapter derives mainly from the choice of appropriate stimulus conditions that reliably elicit the target physiological change, the emphases on transfering training directly to the stress, and a suitable design for evaluating the physiological and subjective effects of the biofeedback training.

The demonstrated consequences of physiological change for emotional arousal are also consistent with peripheral conceptions of emotion derived from the James-Lange Theory (Fehr & Stern, 1970; James, 1890). The finding that individual differences in cardiac awareness can be important in the obtained pain perception effects supports this interpretation. However, the degree to which individual differences in autonomic awareness plays a critical role in stress response requires further experimentation.

The importance of autonomic feedback is also reinforced by the common experience of an association between anxiety, fear, and other reactions to stress and an increase in heart rate or physiological arousal (see Harris & Katkin, 1975). The occurrence of such physiological arousal may serve as a cue for emotionality. In contrast, reduced heart rate or other autonomic deactivation may be less compatible with emotionality. Systematic desensitization is based on this relationship (Wolpe, 1958).

Biofeedback training for individually-relevant physiological changes occurring in association with stressful stimuli has served as a behavioral strategy for changing anxiety and fear reactions. Several reports of clinical research have appeared which utilize these kinds of procedures (Blanchard & Abel, 1976; Gatchel & Proctor, 1976; Nunes & Marks, 1975; Prigitano & Johnson, 1972). More recent systematic research on the use of heart rate biofeedback in reducing anxiety suggests that expectancies and other non-specific placebo effects probably have a major influence on the therapeutic benefits obtained with these methods (see Gatchel, 1979). The research described in this chapter has only touched upon the many complex issues that may be involved in clinical situations. Moreover, it is difficult to generalize from the reported laboratory research on physical stressors to situations involving psychological stressors or clinical pain. Nonetheless, this research provides some systematic experimental support for further research

and clinical applications on the use of biofeedback in the management of stress reactions. The research calls attention to the importance of appropriate instructions, the need to bring the critical environmental stimuli into the therapeutic situation, the significance of developing an active coping skill to facilitate transfer of training, and the potential role of individual differences in autonomic awareness in bringing about desired benefits. Hopefully, the methods and research findings described in this chapter will lead to further productive basic research on the psychophysiology of stress, pain, and anxiety and more effective clinical approaches to their management.

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Table 1[a]

Comparison of Mean Pain Ratings and Mean Heart Rate Effects During the Second Cold Pressor Test

Group	Pain Rating[c]	Heart Rate Indices[b] (bpm) 1	2	3
Increase-Feedback	6.7	88.6	11.5	11.6
Increase-No Feedback	6.1	75.7	4.4	4.1
Habituation-Control	6.3	74.3	0.2	4.1
Decrease-No Feedback	5.4	73.5	0.1	0.5
Decrease-Feedback	4.0	70.2	-6.0	-3.3

[a]From Victor, R., Mainardi, J. A., & Shapiro, D. Effect of biofeedback and voluntary control procedures on heart rate and per eption of pain during the cold pressor test. Psychosomatic Medicine, 1978, 40, 216-225.

[b] 1. Mean heart rate during 30 sec of Cold Pressor 2.

2. Mean heart rate during 30 sec of Cold Pressor 2 minus mean heart rate during 30 sec of Cold Pressor 1.

3. Mean heart rate in 30 sec of Cold Pressor 2 minus mean heart rate in prior 5-sec base period, subtracting out parallel change in Cold Pressor 1.

[c] 1 = no pain; 10 = unbearably painful

Table 2

Intensity and Affective Pain Descriptors and

Psychophysically Scaled Values

Intensity Scale		Affective Scale	
Extremely intense	60.2	Excruciating	30.2
Very intense	47.0	Intolerable	23.5
Very strong	34.4	Unbearable	20.7
Intense	33.8	Agonizing	19.0
Strong	25.4	Horrible	16.0
Slightly intense	21.1	Dreadful	14.6
Barely strong	16.1	Frightful	13.1
Moderate	11.2	Awful	11.6
Slightly moderate	9.0	Miserable	10.9
Very moderate	8.9	Oppressive	10.7
Mild	5.1	Distressing	6.4
Very mild	3.3	Uncomfortable	4.0
Weak	2.6	Unpleasant	3.8
Very weak	1.3	Distracting	3.1
Extremely weak	0.7	Bearable	2.9

Table 3

Heart Period and Pain Rating Data

(Reeves et al., 1980)

Group	Heart Period			Pain Rating[d]	
	Feedback Trials[a]	Feedback Trials[b]	Cold Pressor[c]	Verbal Pain Rating	Visual Analog Scale
I-I	+67	808.	+48.	+0.9	+1.39
D-D	-28.	871.	-42.	-1.0	-1.13
I-D	-15.	858.	-16.	-0.1	+0.21
D-I	-20.	859.	-55.	-0.3	+0.66

[a]Difference in mean heart period in msec between 15-sec pretrial and 45-sec trial period (positive number means a decrease in interbeat interval or increase in heart rate).

[b]Mean heart period in msec collapsed across trials.

[c]Difference in mean heart period in msec between Cold Pressor 1 and Cold Pressor 2 (positive number means a decrease in interbeat interval or increase in heart rate).

[d]Difference in pain rating scales between Cold Pressor 1 and Cold Pressor 2 (positive sign means increase in pain).